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Atrial fibrillation in the Wolff-Parkinson-White syndrome

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In atrial fibrillation complicating the Wolff-Parkinson-White (WPW) syndrome the incessant impulses can be conducted via the bypass to unprotected ventricular muscle and thereby induce ventricular fibrillation. We have observed 10 patients with WPW syndrome who had documented paroxysms of atrial fibrillation. During atrial fibrillation, total anomalous conduction was present in 8, and exclusive atrioventricular nodal conduction in 2. An additional patient who developed atrial fibrillation during an electrophysiological study showed conduction down the anomalous pathway. Four patients were classified as having left-sided bypasses (type A), 4 right-sided (type B), and 2 indeterminate (probably postero-septal). These patients had suffered one or more attacks of atrial fibrillation during a period of up to 16 years before referral; one had suffered a single attack only, the others as many as 12. Only one died suddenly (there was no necropsy); another died of an unrelated cause; the remainder are well, some on appropriate antiarrhythmic therapy. With intracardiac studies we were able to define the functional properties of the components of the anterograde pathway, which include the atria, the bypass, and the ventricles, and relate these to the maximum possible ventricular rate. It is clear that liability to ventricular fibrillation is dependent not only on the functional refractory period of the bypass and the conduction velocity down it, but also on properties of the atria and ventricles, as well as on factors at present unknown. Studies on cases like these may be deceptive because the patients at greatest risk may well have died in their first attack. Consideration of the possible risks of developing atrial fibrillation and of the responses to this rhythm merit greater attention in the electrophysiological assessment of patients with WPW syndrome.

Pathology of conducting system in sinoatrial disease

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In recent years there has been increased interest in the various clinical syndromes associated with sinoatrial disease and sinus bradycardia, and several large series of cases have been reported. However, the aetiology of the condition remains unexplained and there are few reports of pathological studies of the sinus node and conducting system. Indeed, search of the published papers has produced only 14 cases. Arteriosclerosis was a usual finding but detailed examination of the vascular supply to the sinus node was only described in a few.

This paper describes the findings in 8 patients dying with sinus node disease. All but one were already known to the Devon Heart Block and Bradycardia Survey, and 5 had been studied in detail during life and followed for 5 or more years.

The histology of the sinoatrial node, His bundle, and other relevant areas was examined by serial sectioning, and where possible the blood supply to these specialized areas has been visualized by postmortem coronary angiography, x-rays being taken onto a fine grain industrial x-ray film and xeroradiography plates. Gross histological abnormalities were common in the sinoatrial node, but the vasculature in general was well maintained.

Comparison of mexiletine and procainamide in prevention of recurrent ventricular arrhythmias after acute myocardial infarction

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Mexiletine and procainamide were compared with placebo in 60 male patients who had sustained a myocardial infarction within the previous 48 hours and had required lignocaine for ventricular tachycardia, R on T, multiform, or close coupled ventricular ectopic beats. The drugs were evaluated by continuous 24-hour recordings of the electrocardiogram on two occasions.

Procainamide was given as 500 mg 4-hourly and mexiletine as 250 mg 8-hourly with corresponding placebo regimens. Though only 35 per cent of patients receiving procainamide achieved standard therapeutic blood levels, compared with 95 per cent on mexiletine, these two drugs showed an equally significant ($P < 0.05$) antiarrhythmic effect compared with a placebo.

Side effects were infrequent in the mexiletine group. Two patients receiving procainamide were shown to be ANF positive.

These results support the use of antiarrhythmic agents after lignocaine in the management of myocardial infarction, and mexiletine has the advantage of less frequent administration.

Effect of lignocaine on mechanisms of dysrhythmias in myocardial infarction

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During perfusion in a tissue-bath, intracellular action potentials were recorded from portions of endocardium removed from the left ventricle of normal dogs, and from dogs 1 to 3 days after ligation of a coronary artery. In infarcted preparations the deep ventricular muscle was histologically and electrophysiologically dead, but pathologically-altered Purkinje fibres survived in the region of the infarct.

Lignocaine hydrochloride (2.5–10.0 μg/ml) did not increase the resting membrane potential of damaged areas, and further reduced the maximum rate of depolarization and slowed conduction in the Purkinje tissue.

Abnormal pacemaker activity continued in damaged areas despite perfusion with a high concentration (4.68 μg/ml). 10.0 μg/ml was usually ineffective.

Cells surviving around the infarct had long action potential durations; lignocaine did not restore these to normal. In non-infarcted tissue adjacent to the infarct, action potentials were short, and re-entry of close-coupled premature stimuli occurred before drug perfusion. These abnormal responses usually increased after perfusion with lignocaine.

Failure to abolish abnormal pacemaker activity, slowing of conduction in damaged areas, and increased re-entry of premature action potentials explain the reported failure of the drug to control ventricular arrhythmias in patients after infarction.

Measurement of postoperative pericardial pressure in man

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A solid-state low drift catheter tip transducer (3 mm diameter) calibrated at 37° C in saline, was introduced into the pericardial space behind the left ventricle in patients undergoing cardiopulmonary bypass, and left *in situ* for up to 10 days. Electrocardiogram and external phonocardiogram were also recorded, and left ventricular pressure measured with a solid state catheter tip transducer for the first 12 to 14 hours. Pericardial pressure traces were reproducible, and demonstrated that maximum pressure occurred immediately before ventricular systole. Pressure fell from the onset of isovolumic contraction and throughout ejection, reaching a nadir in the early diastolic rapid filling phase as ventricular pressure was falling. Fluctuations in intrapericardial pressure during spontaneous respiration or artificial ventilation were small, suggesting that concomitant diastolic pressure changes in the left ventricle were caused mainly by alterations in diastolic filling. Relative ventricular and pericardial distensibility could be estimated from the ratio of rates of increase of diastolic pressure with time on the two traces. Since mean pericardial pressures of up to 15 mmHg (2.0 kPa) with respect to atmospheric were recorded, the use of postoperative measurements of filling pressure to assess ventricular function may require reconsideration.

Haemodynamics of cardiac restriction and tamponade

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Haemodynamic data are necessary for differential diagnosis in cardiac restriction and to elucidate the pathophysiology. Accordingly, cardiac catheterization and quantitative left ventriculography were performed on 3 patients with constrictive pericarditis, 3 with cardiac amyloidosis, and 3 with cardiac tamponade. End-diastolic pressure was raised in both ventricles in all patients. Left ventricular end-diastolic volume was reduced in all patients except one who had mitral incompetence.

In constrictive pericarditis and cardiac amyloidosis ventricular filling was confined to early diastole and occurred when left ventricular pressure was still falling. A plateau of volume and pressure characterized mid and late diastole. In cardiac tamponade, the left ventricle filled throughout diastole and the diastolic dip and plateau were absent. In cardiac amyloidosis, left ventricular diastolic pressure exceeded right, but these pressures were equal in constrictive pericarditis and in cardiac tamponade.

Diastolic compliance was reduced in all patients and the ventricular filling mechanism differed between restriction and tamponade. Contractility was normal in tamponade. Both in constrictive pericarditis and in cardiac amyloidosis, ejection and isovolumic contractility parameters were variable and, therefore, did not separate myocardial from pericardial restriction. Comparison of left and right heart filling pressures remains the most useful tool to distinguish between restrictive cardiomyopathy and constrictive pericarditis.

Enzyme studies on myocardial biopsies in congestive cardiomyopathy[1]

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The development of the endomyocardial biopsy technique has made it possible to obtain tissue for both diag-

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nostic and investigative purposes. In an attempt to look for biochemical changes in cardiomyopathy, marker enzymes of the various subcellular organelles have been assayed in both control and myopathic human tissue obtained during routine cardiac catheterization. The following enzymes (organelles between parentheses) were measured using highly sensitive assay techniques: Na^+, $K,^+$-activated Mg^{2+} dependent ATPase, 5'nucleotidase (plasma membrane); Mg^{2+}-activated ATPase, glutamate and malate dehydrogenase, monoamine oxidase (mitochondria); acid phosphatase, N-acetyl-β-glucosaminidase (lysosomes); neutral α-glucosidase (microsomes); azide-resistant Ca^{2+}-activated ATPase (myofibrils); lactate dehydrogenase (cytosol). Using tissue obtained from both ventricles no significant differences were noted for the lysosomal, microsomal, or plasma membrane marker enzymes between right or left ventricles in either control or myopathic tissue. Reduced levels of the mitochondrial dehydrogenases and of the myofibril ATPase were found in tissue obtained from patients with poor left ventricular function, defined by reduced KVmax values during sinus rhythm, with little postectopic potentiation. In contrast, threefold increases in lactate dehydrogenase were found in the myopathic tissue.

It is suggested that in congestive cardiomyopathy diminished myofibrillar contractility is associated with impairment of mitochondrial energy production and consequently with enhanced anaerobic glycolysis.

Labilization and stabilization of lysosomes in anoxic guinea-pig heart[1]

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Isolated perfused guinea-pig hearts were used to study the effects of anoxia on myocardial lysosomes. Lysosome stability was assessed by measuring latent and sedimentable acid hydrolase activities in myocardial extracts. Control aerobically perfused hearts showed unchanged lysosomal stability for at least 6 hours of perfusion. In contrast, anaerobically perfused hearts showed a striking decrease in lysosomal stability after 30 minutes of perfusion. This progressed with the duration of anoxia.

Addition of glucose (11.1 mmol) to the perfusion medium almost completely protected the lysosomes from anoxic damage. Mannitol, a non-metabolized carbohydrate, afforded only limited protection suggesting that the effect of glucose was chiefly caused by its metabolic activity. Methyl prednisolone (10^{-5}M) and chloroquine (10^{-7}mmol) also protected the myocardial lysosomes against anoxic labilization. These studies suggest that lysosomes may be implicated in the pathogenesis of anoxic myocardial damage and necrosis, and indicate that lysosomal stabilizing agents may have a beneficial effect.

[1] This work was supported by the British Heart Foundation.

Diagnosis and prognosis of bicuspid non-stenotic aortic valve

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Congenitally bicuspid aortic valves have been recognized by pathologists for many years as the commonest congenital anomaly of the heart, and the complications of stenosis, regurgitation, and bacterial endocarditis are well known. It has been suspected that an isolated early systolic sound might be a means of diagnosing clinically the presence of a non-stenotic, bicuspid aortic valve.

Fifty-one subjects with an aortic ejection sound, with or without a trivial ejection systolic murmur and without other evidence of heart disease, have been followed for 5 to 20 years. Five died and necropsy findings were available in 4. A congenitally bicuspid valve was demonstrated in each case; in one associated with recurrent bacterial endocarditis, and in 3 with an otherwise normal valve and death from other causes. Three patients developed bacterial endocarditis followed in 2 by aortic regurgitation. Three developed slight aortic regurgitation without infection. In 2 subjects, who over a period of 13 and 14 years, developed calcific aortic stenosis, requiring valve replacement, a bicuspid aortic valve was found at surgery. In the remaining 34 subjects, the ejection sound, with or without a soft ejection systolic murmur, remains the only physical sign. Observations made from echo and phonocardiograms and from the carotid pulse, however, lead us to believe that they too have a bicuspid aortic valve, and these signs will be described.

Morphogenesis and nomenclature of univentricular hearts

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There is considerable confusion surrounding the terminology of hearts which apparently possess a single ventricular chamber. The terms 'single ventricle', 'common ventricle', or 'double inlet left ventricle' are presently used in different fashions and with different meanings by different investigators. On the basis of our morphological and histopathological studies of a large series of such hearts, we suggest that they can be classified, and the disposition of conducting tissue predicted on the basis of the presence or absence of one or more of three embryonic ridges. These ridges are the right and left bulboventricular (BV) ridges, which separate the primitive ventricle from the primitive bulbus, and the interventricular (IV) ridge which septates the primitive ventricle. When both right and left BV ridges persist in the absence of the IV ridge, the bulbus persists as an outlet chamber. We suggest this anomaly should be termed primitive ventricle with outlet chamber. When only the

left BV ridge persists, bulbus and ventricle form a common chamber which we suggest should be termed primitive ventricle with absent outlet chamber. Our studies also indicate that both those anomalies can exist with either normally positioned or transposed arteries, or with atresia (or failure of development) of an atrioventricular orifice.

Clinical, angiographic, and morphogenetic correlations in tetralogy of Fallot

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It has been long established that patients with tetralogy of Fallot present with varying symptomatology. It has also been established that in morphological terms the anomaly represents a spectrum of disturbances based on rotation of the conus and anterior deviation of the conus septum. We have studied the mode of presentation of infants subsequently found to have tetralogy of Fallot, and compared their angiograms with pathological specimens. Four basic patterns of presentation could be distinguished, though intermediates were observed between each group. In the patients presenting with severe cyanosis in the neonatal period, the major feature was severe anterior deviation of the conus septum with little conal rotation, so that the conus septum was visualized on lateral angiograms. In the groups presenting with episodic loss of consciousness or gradually increasing cyanosis, both conal rotation and deviation of the conus septum were present. The conus septum was not well visualized in either frontal or lateral angiograms. The progression of symptoms corresponded to increasing right ventricular hypertrophy. The fourth group presented initially with dyspnoea and plethora on chest *x*-ray. At this stage conal rotation was the major feature, with the great arteries orientated in side-by-side fashion (PA projection). Reversal of the shunt was referable to increasing infundibular hypertrophy which produced pulmonary stenosis.

Use of right atrium to pulmonary artery valved conduit for 'correction' of single ventricle of hypoplastic right heart syndrome

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Four patients with single ventricle and pulmonary stenosis were treated by using a valved conduit from the right atrium to the main pulmonary artery. The mobilized patient's own pulmonary valve was used in 1 patient and fresh adult sized aortic homograft in the remaining 3. Two additional unstented homografts were inserted at the entrance of both superior and inferior venae cavae in 1 patient. A pericardial baffle was used to direct pulmonary venous blood to both atrioventricular valves in 3 and the tricuspid valve was oversewn in 1. Transposition of the great arteries was present in 2 and hypoplasia of the tricuspid valve in 1. The age at operation was 4 months to 9 years. All patients were severely incapacitated with arterial oxygen saturation varying from 28 to 60 per cent. One infant died 24 hours after operation; the remaining 3 are alive and well 6 to 12 months after operation, with excellent symptomatic improvement.

Repeat cardiac catheterization performed in the 3 survivors showed a mean right atrial pressure varying from 4 to 10 mmHg (0.5 to 1.3 kPa) and a prominent right atrial A wave, measuring 8 to 14 mmHg (1.1 to 1.9 kPa). The arterial oxygen saturation was above 93 per cent in all 3.

Tricuspid atresia – new hope with radical palliative surgery

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Since 1972, 8 patients with tricuspid atresia aged 6 to 20 years have had closure of the interatrial communication and anastomosis of the right atrial appendage to the main pulmonary artery or right ventricular outflow using an intermediate conduit containing an aortic homograft valve. Valves were not put in the superior vena cava and only two had a homograft valve put in the orifice of the inferior vena cava.

There have been 3 operative deaths caused by wrong selection of patients in 2; 1 died with low output and atrial fibrillation, having had preoperative left ventricular failure from a large shunt, and another with associated transposition had subaortic obstruction from a closing ventricular septal defect. The other died from hypoxia after early reopening of the atrial septal defect caused by broken prolene sutures.

The 5 survivors, assessed 6 months to 2 years later, are leading normal lives without symptoms, with normal heart size, no change on the electrocardiogram, and slight hepatomegaly in 2 with normal liver function tests. Cardiac catheterization has shown right atrial pressures 12 to 15 mmHg (1.6 to 2.0 kPa) with 1 to 3 mmHg (0.1 to 0.4 kPa) gradients across the conduit valve on the A wave; normal cardiac output at rest and on effort was found in one. Pulmonary valve closure was recorded in one after atrial systole. Important lowering of blood pressure occurred with atrial fibrillation and nodal dysrhythmias.

Although a palliative correction, if long-term success is maintained, it offers new hope for certain patients with tricuspid atresia and common ventricle. There may be a place for this earlier in the natural history before irreversible damage to the left ventricle, distortion of pulmonary arteries from previous shunts, or underdevelopment of the pulmonary valve and artery after critical diminution in the size of the ventricular septal defect.

Echocardiography in evaluation of congenital mitral valve disease in infants and children

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Congenital mitral valve disease, particularly when associated with other cardiovascular malformations, may be difficult to recognize. The purpose of this study was to assess the usefulness of echocardiography in evaluating mitral valve function in infants and children with suspected heart disease.

Nine children with confirmed congenital mitral stenosis were studied. In 4 of them mitral stenosis had not been suspected clinically, and in 2 cardiac catheterization had also not been diagnostic of mitral stenosis. A reduced rate of closure of the anterior leaflet of the mitral valve during diastole was noted in all, but a similar finding was observed in some patients with severe pulmonary hypertension, obstructive cardiomyopathy, or aortic stenosis. Patients with true mitral stenosis were distinguished by identification of abnormal anterior movement of the posterior leaflet of the mitral valve in diastole.

Forty-two patients with clinical or angiographic evidence of mitral regurgitation were studied. In 17, echocardiography showed evidence of prolapsing anterior or posterior leaflets of the mitral valve. In 8 with left ventricular outflow tract obstructive cardiomyopathy, echocardiography showed abnormal midsystolic movement of both the anterior and posterior leaflets of the mitral valve. In 17 with complex congenital heart disease associated with mitral regurgitation, precise definition of the echocardiographic anatomy and the relation of the mitral valve was usually possible, and specific features characterized defects of the endocardial cushions and mitral regurgitation in corrected transposition of the great arteries.

Echocardiography has thus been shown to be of value not only in identifying unsuspected mitral valve disease, but also in amplifying the findings of cardiac catheterization.

Instantaneous mitral valve velocity and its relation to ventricular wall movement in man

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Echocardiograms of the anterior mitral valve leaflet and the ventricular cavity were recorded at 100 mm/s, digitized, and their first derivatives computed. Instantaneous measures of mitral valve velocity, left ventricular dimension, and their rates of change were thus obtained.

In patients with normal left ventricular contraction judged angiographically, the onset of mitral valve and wall movement occurred within 0.03 s (SD = 0.02 s) of each other. Peak opening velocity was reached within 0.05 s (SD = 0.01 s) and coincided with the peak rate of wall movement ($\pm$0.03 s). The mean acceleration of mitral valve opening was 0.61 $\pm$ 0.24 *g*. The velocity of mid-diastolic closure increased as that of wall movement was falling, and the peak closure rate corresponded closely with a sharp reduction in the velocity of wall movement in mid-diastole. Peak closure rates were considerably greater than those measured by routine manual methods on the same records.

In patients with incoordinate wall movement judged angiographically, a significant increase in ventricular dimension occurred before mitral valve opening, and the normal relation between mitral valve and wall movement was lost.

This technique thus allows unambiguous measurement of mitral valve movement and demonstrates its close relation to normal and abnormal patterns of left ventricular filling.

Nitroglycerin and nitroglycerin-phenylephrine-induced reduction in ischaemia during acute myocardial infarction in man

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Nitroglycerin reduces ischaemia during acute myocardial infarction in dogs, an effect potentiated when drug-induced hypotension and tachycardia are prevented with an alpha-adrenergic agonist such as phenylephrine. To determine the effectiveness of nitroglycerin/phenylephrine therapy in man, 10 patients were evaluated early during acute myocardial infarction by summing ST segment abnormalities ($\sum$ST) from 35 praecordial electrodes. Pulmonary artery wedge pressure was raised (14 to 35 mmHg (1.9 to 4.7 kPa)) in 4 patients. Before treatment $\sum$ST changed spontaneously by $<0.5\%/5$ minutes over 15 to 90 minutes. In contrast, therapy with nitroglycerin plus phenylephrine caused obvious improvement in $\sum$ST in all 10 patients (mean reduction 2 mV; 5%/5 minutes, $P<0.01$). Eight patients initially received sublingual nitroglycerin alone (0.3 mg/minute for 5 minutes); 5 minutes after therapy $\sum$ST improved in 7 (mean reduction 0.95 mV; 12%/5 minutes), but worsened in 1 in whom nitroglycerin caused mild hypotension and pronounced tachycardia. When arterial pressure fall was reversed with intravenous phenylephrine, $\sum$ST decreased in all 8 patients (mean 1.5 mV; 5%/5 minutes). In 2 other patients combination therapy alone was given; $\sum$ST decreased considerably in both. We conclude that nitroglycerin/phenylephrine therapy may be of value early during acute myocardial infarction in man, since nitroglycerin alone usually reduces ischaemia, and reduction of nitroglycerin-induced hypotension and tachycardia with phenylephrine potentiates the benefits and reverses the occasional detrimental effects.

Isotope scanning in the heart: clinical results

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The technique of isotope scanning of the heart, using both ^{129}caesium and ^{99m}technetium-labelled human serum albumin intravenously, was described to the Society in 1974. The caesium displays viable, perfused myocardium and the technetium the cardiac blood pool. A subtraction technique enhances the myocardial image and independent visualization of the left ventricular cavity and myocardium becomes possible.

Fifty patients, including those with ischaemic, valvular, and primary myocardial heart disease and with non-cardiac chest pain, have been studied. The results have been correlated with electrocardiographic, radiographic, and echocardiographic data.

Normally, significant caesium uptake is seen only in the myocardium of the left ventricle. In the presence of adequate tissue perfusion and uptake three views permit an assessment of left ventricular dimensions. Characteristic appearances are seen in left ventricular hypertrophy, left ventricular dilatation, and hypertrophic obstructive cardiomyopathy.

In patients with myocardial infarction the non-viable area can be visualized. The area of defective uptake correlates with electrocardiographic localization.

Dual isotope scanning thus provides a non-invasive method of estimating the left ventricular cavity size and the mass and viability of the left ventricular myocardium.

Postpartum hypertension and aetiology of peripartum cardiac failure

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Peripartum cardiac failure is an uncommon syndrome of cardiac failure of unknown aetiology, occurring in relation to childbirth, often associated with transient hypertension, and commonest after twin deliveries and in grand multiparae. Most patients have been American Negroes: the syndrome is also common in Korea and in Africa, and exceedingly common among the Hausa women of northern Nigeria. Within the Hausa area, peripartum cardiac failure is closely related to the intensity of local puerperal customs, including salt-loading, and there is a pronounced seasonal variation.

Symptomless postpartum hypertension, resolving spontaneously after a few weeks or months, has been noted in about 15 per cent of Negroes in America and in West Africa. It has not been described in other races, but it is common in Korea, and exceedingly common among Hausa women in northern Nigeria, particularly after twin deliveries, and in grand multiparae, and it has a very similar seasonal variation and time-course to those of peripartum cardiac failure.

Although not all patients with peripartum cardiac failure are hypertensive on admission, the obvious correlations between symptomless postpartum hypertension and symptomatic peripartum cardiac failure suggest that the symptoms of this cardiomyopathy may be precipitated mainly by an acute pressure overload in people environmentally and/or possibly genetically predisposed to postpartum hypertension, though volume overload may also be implicated.

Survival in the Andes (sound colour film)

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A sound colour film lasting 40 minutes was made on a visit to Peru in 1973 by the authors. It describes the pathology of certain cardiopulmonary adaptations to life at high altitude. The body build of the Quechua Indian is described and aspects of the daily life of the Indians illustrated. Reference is made to the ultrastructure of high altitude pulmonary oedema. The changes in the muscular and elastic pulmonary arteries associated with the development of pulmonary hypertension of high altitude are discussed. The film describes the histological and electron microscopical features of the carotid body which occur in man and animals with the chronic hypoxia of diminished barometric pressure. Finally cases of Monge's disease, sometimes referred to as chronic mountain sickness, are illustrated and the controversial nature of this condition discussed.